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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/446,996	12/30/1999	JOHANNES CHRISTIANUS VAN GROENINGHEN	49477(1958)	3246

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EXAMINER	
BORGEEST, CHRISTINA M	
ART UNIT	PAPER NUMBER
1649	

SHORTENED STATUTORY PERIOD OF RESPONSE	MAIL DATE	DELIVERY MODE
3 MONTHS	02/26/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

Office Action Summary	Application No.	Applicant(s)	
	09/446,996	VAN GROENINGHEN, JOHANNES CHRISTIANUS	
	Examiner	Art Unit	
	Christina Borgeest	1649	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *04 August 2006*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-9,12 and 14-20* is/are pending in the application.

4a) Of the above claim(s) *1-9 and 12* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *14-20* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *23 August 2006*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Response to Amendment

The amendment submitted 4 August 2006 is acknowledged. Claims 1-9, 12 are withdrawn. Claims 10-11, 13 are cancelled. Claims 14-17 are amended. Claims 18-20 are new. Claims 14-20 are under examination.

The text of those sections of 35 U.S.C. not included in this action can be found in a prior office action mailed 29 June 2001.

Objections Withdrawn

The objection to the specification with regard to compounds described as antagonists in Table 1 are referred to agonists on p. 10, line 3 is withdrawn in response to Applicants' correction of the specification in order to correct an obvious error.

Information Disclosure Statement

The objection to the information disclosure statement filed 17 August 2005 for failing to comply with the provisions of 37 CFR 1.97, 1.98 and MPEP § 609 is withdrawn in response to Applicants providing copies of the references.

Rejections Maintained/New rejections

Claim Rejections - 35 USC § 112, first paragraph

The rejection of claims 14-17 under 35 U.S.C. 112, first paragraph, is maintained in part. In addition, new claims 18-20 are also rejected under 35 U.S.C. 112, first paragraph. Claims 14-20 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for decreasing cellular replication of a GnRH-receptor positive malignant melanoma comprising administering a GnRH agonist, or optionally, a GnRH agonist in combination with a cytotoxic substance, does not reasonably provide enablement for the methods as broadly claimed. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention commensurate in scope with these claims. There are many factors to be considered when determining whether there is sufficient evidence to support a determination that a disclosure does not satisfy the enablement requirement and whether any necessary experimentation is "undue." (See In re Wands, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404 Fed. Cir. 1988) These factors include, but are not limited to: (a) the breadth of the claims; (b) the nature of the invention; (c) the state of the prior art; (d) the level of one of ordinary skill; (e) the level of predictability in the art; (f) the amount of direction provided by the inventor; (g) the existence of working examples; and (h) the quantity of experimentation needed to make or use the invention based on the content of the disclosure. Briefly, treatment of cancers other than melanoma are not enabled by the claimed methods because as will be explained in greater detail below, there are no teachings in the instant specification or the prior art to

support enablement for the treatment of brain cancer (claim 14 encompasses any type of brain-tissue derived cancer), Ewing sarcoma; Kaposi sarcoma, Glioblastoma multiforme, medulloblastoma, pinealoma, neuroblastoma, craniopharyngeoma, meningeoma, chordoma (recited in claims 14, 16, 19). The state of the prior art, the level of predictability in the art, the amount of direction provided by the inventor, the existence of working examples and the quantity of experimentation needed to make or use the invention based on the content of the disclosure are all factors that the Examiner must consider when deciding whether a case is enabled. In this case, the lack of disclosure and working examples coupled with the high level of unpredictability in the art with respect to the treatment of the recited cancers with a GnRH agonist or antagonist leads the Examiner to the conclusion that the claimed methods are not fully enabled. In addition, as is discussed in greater detail below, there is no support in the prior art for the use of GnRH antagonists in the claimed methods; in other words the preponderance of the evidence does not support full enablement of the claims.

Applicants' arguments are paraphrased below (single-spaced) with the Examiner's response (double-spaced) immediately following each argument.

Applicants' argue at p. 9, 1st full paragraph that as amended, independent claims 14-16 are drawn to methods for decreasing cellular replication of GnRH-receptor positive tumors using GnRH agonistic or antagonistic analogues, and state that tumors originating in the brain, nervous system or meninges of the brain, including glioblastoma multiforme (GBM), glioma, meningeoma and chordoma, are GnRH-receptor positive. Applicants further state that Example 14 describes inhibition of melanoma cell proliferation by a GnRH agonist (Triptorelin) or a GnRH antagonist (Antide), providing support for the presence of GnRH receptors on human malignant melanoma and that support for the presence of GnRH receptors on Kaposi sarcoma may be found, for example, at page 6, lines 6-7 of the specification. Finally, Applicants state that further evidence of the presence of GnRH-receptors in melanoma or Kaposi sarcoma has been provided in related U.S. Application Serial No. 10/327,621 in the form of a declaration

executed by the inventor, Dr. Johannes C. van Groeninghen, according to 37 C.F.R. § 1.132 (copy provided herewith). Applicants cite numerous articles which teach that GnRH receptors are present in various types of cancer.

These arguments have been fully considered but are not found persuasive for the following reasons. The declaration under 37 CFR 1.132 filed with respect to Application 10/327,621 is insufficient to overcome the rejection of claims based upon 35 U.S.C. 112, first paragraph as set forth in the last Office action because no copy of the declaration has been provided, and it is not of record in the instant case. Applicant states that a copy has been provided in the instant case, but none can be found, thus any evidence contained therein cannot be considered with respect to the instant case. Furthermore, Moretti et al., J. Clin. Endocrino. Metab., 2002, 87(8):3791-7 (cited in Applicants' 1449 form from 17 August 2005) states at p. 3795, right column, 1st paragraph that "LHRH [another term in the art for GnRH] receptor activation by an exogenous LHRH agonist brings about a significant decrease of tumor cell proliferation. The inhibitory effect of the LHRH agonist is specific because it is ***counteracted by the simultaneous treatment of the cells with a LHRH antagonist***." (Emphasis added). This reference teaches that the tumor inhibiting effects of the LHRH (aka GnRH) agonist can be reversed by antagonists, thus does not support enablement for the claimed methods in their full scope. Furthermore, Moretti go on to say at p. 3795, right column, end of the 1st paragraph, "The activation of the inhibitory system, through use of the ***LHRH agonists*** may reduce tumor growth and interfere with the positive effect of the mitogenic factors." Thus again, Moretti et al. underscore that only the agonists activated tumor inhibition. Furthemore, Groeninghen et al. conclude in a research letter

in the Lancet (1998; 352: 372-373--cited in Applicants' 1449 form from 17 August 2005) that "[the] present confirmation of an autocrine LHRH-regulated growth, implicating tumor self-production of LHRH receptors, could be important for nervous system tumor diagnosis and therapy. LHRH receptors might serve as a prognostic marker for early tumor recurrence, especially for glioma. Further clinical research is needed to establish nervous system tumors as indicators for the use of analogues of LHRH, which block LHRH function." This indicates that further research is needed to determine whether the claimed methods could work, and as the quantity of experimentation needed to make or use the invention based on the content of the disclosure is one of the factors in deciding enablement, this statement in Groeninghen et al. does not support enablement.

Applicant argues at p. 10, 1st paragraph that the USPTO has made it clear that an in vitro or in vivo animal model example in the specification constitutes a "working example" if that example correlates with a disclosed or claimed method invention, and a rigorous or an invariable exact correlation is not required. M.P.E.P. 2164.02, citing Cross v. Iizuka, 753 F.2d 1040, 1050, 224 USPQ 739, 747 (Fed. Cir. 1985). Applicant provides evidence in the form of a string of references, both before and after the filing date of the instant application to show that in vitro inhibition of cell proliferation by a GnRH agonist or antagonist correlates with in vivo anti-cancer activity:

(1) In Pinski et al., Int. J. Cancer., 1994, 59(1): 51 (abstract only; copy provided), it was shown that a LHRH (also referred to as GnRH) antagonistic analogue and a LHRH agonistic analogue not only significantly prolonged the mean survival time of rats bearing prostatic adenocarcinoma, but also suppressed proliferation in cell cultures, respectively. Human clinical evidence that treatment with a LHRH agonist led to reduction of prostate tumor mass and metastases may be found in Damyanov et al., Eur. Urol., 2001, 40(4):474-6 (abstract only; copy provided).

(2) Kim et al., Gynecol. Oncol., 1999, 74(2): 170-180 (abstract only; copy provided), showed that continuous exposure of two human ovarian cancer cell lines and xenografts to a GnRH agonist resulted in growth inhibition of cancer cells in a dose- and time-dependent manner. A phase II clinical study of showed that Cetrorelix, a GnRH antagonist, has activity against ovarian cancer in human patients. See Verschraegen et al., 2003, 90(3):552-559 (abstract only; copy provided).

(3) In Vincze et al., J. Cancer Res. Clin. Oncol., 1994, 120(10): 578 (abstract only; copy provided), a GnRH antagonistic analogue (MI-1544) induced a significant decrease in cell numbers of human breast cancer cell lines in vitro, and inhibited the growth of xenografts in vivo. Further evidence of in vivo antiproliferative effect of a GnRH agonist and a GnRH antagonist on breast cancer may be found in Yano et al., Breast Cancer Res. Treat., 1992, 21 (1):35-45 (abstract only; copy provided), in which tumor volume was significantly suppressed by both the GnRH agonist and the GnRH antagonist in nude mice. Clinical studies indicate that goserelin, a GnRH agonist, is effective for chemotherapy for early breast cancer. See Kaufmann et al., Eur. J. Cancer, 2003, 39(12):1711-7 (abstract only; copy provided).

(4) Limonta et al., Frontiers in Neuroendocrinology, 2003, 24:279-295 (page 287—previously provided with the response filed on 08/17/2005, reported inhibition of melanoma cell proliferation both in vitro and in vivo by GnRH agonists.

These arguments have been fully considered but are not found persuasive. The Examiner does not take issue with the fact that an in vitro or in vivo animal model example in the specification constitutes a "working example" if that example correlates with a disclosed or claimed method invention. The issue is, given the evidence cited by Moretti et al. and Groeninghen et al., the claimed methods are not commensurate in scope with the evidence. The claimed methods are drawn to the treatment of brain cancer (claim 14 encompasses any type of brain-tissue derived cancer), Ewing sarcoma; Kaposi sarcoma, malignant melanoma, Glioblastoma multiforme, medulloblastoma, pinealoma, neuroblastoma, craniopharyngeoma, meningeoma, chordoma (claims 14, 16, 19) comprising administration of GnRH agonists or antagonists with or without coadministration of a cytotoxic drug (claim 17 has the further limitation of coadministration of a cytotoxic drug). Moretti et al. teach only that agonists might be useful in the treatment of melanoma and Groeninghen et al. state that further research is needed to establish whether neurological cancers can be treated using GnRH analogs. With respect to the articles cited as evidence by Pinksi et al., Kim et al.

and Vincze et al., all these references deal with the treatment of reproductive cancers, not the cancers recited in the claims, thus again, the evidence does not support the scope of the claimed invention. If the teachings of Pinski et al., Kim et al. and Vincze et al. could provide support for the claimed methods, then by extension, the claimed methods could be rendered obvious by those teachings, which is not the case in the instant application. With regard to Limonta et al., the authors teach that a single GnRH agonist, Goserelin, might have anti-metastatic activity in a melanoma cell line, although the authors are cautious in their language (see p. 287, whole page), which supports the Examiner's position that the claimed methods are not commensurate in scope with what is taught in the specification and the prior art.

Applicants argue at p. 11, 3rd paragraph that the GnRH analogues are well-studied compounds and their mechanism of action is known, namely by binding to the GnRH receptor and affecting downstream biological events such as signal transduction, whereas much less is known about each of the 70,000 compounds screened in the NCI program in the article cited by the Examiner (Shi et al.), and that GnRH agonistic or antagonistic analogues have been tested in a variety of cancer cell lines, including melanoma, breast cancer, ovarian cancer, and prostatic adenocarcinoma, as shown in the references cited in the proceeding paragraphs.

This argument has been fully considered but is not found persuasive, because as noted above, the evidence in the literature and the specification is not commensurate in scope with the claims. The prior art (Moretti et al. and Limonta et al.) only provide enabling support for reducing proliferation in human melanoma cells in culture via administration of an agonist. Applicant's declaration under 37 CFR 1.132, filed with respect to Application 10/327,621, could not properly be considered in this case because no copy has been provided, thus it is not of record in the instant case. Although Applicants show a 15 to 30% inhibition of proliferation in a human melanoma

cell line with the high doses of a GnRH antagonist (although an ***stimulation of growth of 40%*** was seen in the low dose—see p. 25, lines 21- 30 of the instant specification), the preponderance of evidence in the literature suggests that only GnRH agonists reduce proliferation in the melanoma cell lines, thus the evidence is not commensurate in scope with the claims.

Applicants argue at p. 12, 2nd paragraph that although the invention is novel, there is a significant amount of literature describing the use of GnRH agonist or antagonists in treatment of other types of cancers in vivo models.

This argument has been fully considered but is not found persuasive for the following reason, namely the claimed methods of reducing proliferation of (i.e., encompasses treatment) of the conditions recited in the claims are not commensurate in scope with what is known in the literature and what is presented in the specification. To summarize what is written above: the literature teaches the treatment of reproductive cancers with GnRH agonist or antagonists and that proliferation in human melanoma lines might be reduced by GnRH agonists, but that this effect is reversed by GnRH antagonists. As stated above, if the teachings of Pinski et al., Kim et al. and Vincze et al. could provide support for the claimed methods, then by extension, the claimed methods could be rendered obvious by those teachings, which is not the case in the instant application. Finally, the literature teaches that more research is necessary to determine whether any analogs of GnRH can be used to treat nervous system tumors. The specification teaches that proliferation in human melanoma cell lines is reduced 15% - 35% after treatment with a GnRH agonist or antagonist. This evidence is not commensurate in scope with the treatment of any type of tumor.

Applicants indicate at p. 12, 3rd paragraph to p. 13, 1st paragraph that the claims have been amended to recite that the GnRH agonist or antagonist is a GnRH analogue and that a peptide analogue of GnRH would also have these common structural features and therefore be easily identifiable as a GnRH analogue without undue experimentation, and includes such analogues as the well-known compounds such as leuprorelin, buserelin, goserelin, antide ramorelix, cetrorelix, etc (see Table I at page 9 in specification).

This argument has been fully considered but is not found persuasive for the reasons cited above, namely, the claimed methods are not commensurate in scope with the evidence presented in the literature or the specification.

Claim Rejections - 35 USC § 102

Upon reconsideration of the literature and the claims, claims 14 and 19 are rejected under 35 U.S.C. 102(b) as being anticipated by Laue et al. (AJDC. 1985; 139: 1097-1100—cited in Applicants' 1449 form from 17 August 2005). Laue et al. teach the administration of an LHRH agonist analog (LHRHa: D-Trp^6-Pro^9-NEt-LHRH—GnRH is also referred to as LHRH, see specification, p. 1, 2nd paragraph) to patients suffering from optic glioma (see p. 1097, under Patients and Methods and Protocol). Specifically, Laue et al. teaches treatment of children suffering from central precocious puberty and optic glioma. Laue et al. conclude that the LHRH agonist is effective in treating central precocious puberty secondary to optic glioma (pp. 1097 abstract). Although Laue et al. do not specifically teach the administration of LHRHa for the treatment of the glioma, glioma cell proliferation would have been inherently treated by LHRHa, regardless whether this fact was appreciated at the time. While Laue does not expressly teach that the glioma cells express GnRH receptors, this property would reasonably be considered

to be inherent to the cells since the cells originated in brain, as recited in the claims. Furthermore, since LHRHa was administered intravenously and reached to certain serum concentration, the glioma cells would have been exposed to the agonist. Therefore, Laue anticipates the instant claims.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 14, 16, 18, 19 and 20 rejected under 35 U.S.C. 103(a) as being unpatentable over He et al. (1986, Clinical Chemistry, Vol. 32, No. 6, pp. 1159, abstract #542—cited in Applicants' 1449 form from 17 August 2005)). The claims are drawn to a method of decreasing cellular replication of a GnRH-receptor positive tumor of the types recited in claim 14, including malignant melanoma comprising administration of a GnRH agonist or antagonist, said GnRH agonist or GnRH antagonist being a GnRH analog. He et al. teach a method of administering LHRH analogs (i.e., GnRH analogs) to a melanoma cell line, wherein the results showed a decreased cell growth rate. He et al. do not teach administration to a subject, and though the claim does not specifically recite it, a reasonable interpretation of the claim is that in vivo treatment (as opposed to an in vitro assay) is implied. He et al. do teach that their in vitro assay could be used to evaluate GnRH analogs for use in cancer treatment, and they state research clinical

and treatment applications as a goal of their experiment. It would have been obvious to the person of ordinary skill in the art at the time the invention was made to modify the teachings of He et al. by administering GnRH analogs to subjects, because He et al. explicitly state that their LHRH analogs may be "assessed in vitro prior to clinical administration," thus there is a strong suggestion in the prior art document to use the methods in a clinical setting, and the teachings of He et al. are not inconsistent with administration of GnRH analogs to a subject. The person of ordinary skill in the art would have been motivated to administer GnRH analogs to a subject because there is strong motivation in the art to find treatment of cancer and indeed He et al. explicitly state, "Use of [GnRH] analogs for the treatment of cancer represents a new endeavor in basic and clinical research. Also several studies suggest that melanoma is a hormone dependent tumor in some patients..." Furthermore, the person of ordinary skill in the art could have reasonably expected success because He et al. reported a decrease in cell growth, thus it would be reasonable to expect success in a subject. Thus the claims do not contribute anything non-obvious over the prior art.

Double Patenting

The provisional rejection of claims 14-17 on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims of copending Application No. 10/327,621 is maintained for reasons of record, however it is noted that claims 9, 10, 12 and 13-18 of copending Application No. 10/327,621 were recently cancelled, and new claims 35-51 were added, however, the substance of the rejection

remains. ***Claims 14-20 are rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 35-40, 50 and 51 of copending Application No. 10/327,621***. Although the conflicting claims are not identical, they are not patentably distinct from each other because the claims in the '621 Application are drawn to a method for decreasing cellular replication of malignant melanoma, GnRH-positive oat-cell carcinoma, malignant melanoma, Kaposi sarcoma or proliferating glioma, comprising administering to a cell a replication decreasing amount of luteinizing hormone releasing hormone (LHRH) LHRH analogue coupled to a cytotoxic agent, GnRH agonistic analogue, or a GnRH agonist selected from the group consisting of leuprorelin, triptorelin, buserelin, goserelin and a pharmacologically acceptable salt of any thereof, and the instant claims are drawn to a "method for decreasing cellular replication of a tumor originating in one or more of the brain, nervous system or meninges of the brain, wherein the illness is selected from group consisting of Kaposi sarcoma, proliferating glioma, glioblastoma multiforme, medulloblastoma, pinealoma, neuroblastoma, craniopharyngeoma, meningeoma, chordoma, Ewing sarcoma or malignant melanoma comprising administering to a subject a therapeutically effective amount of one or more of a GnRH agonist or GnRH antagonist. The broad claim language in the instant application is encompassed by claims 35-40, 50 and 51 of copending Application No. 10/327,621.

This is a provisional obviousness-type double patenting rejection because the conflicting claims have not in fact been patented.

Applicant's argument at p. 13, last paragraph that the obviousness-type double patenting rejection should be withdrawn because it is the only rejection remaining in the instant application is moot, since there are two remaining grounds of rejection in the instant application.

Conclusion

No claim is allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Christina Borgeest whose telephone number is 571-272-4482. The examiner can normally be reached on 8:00-4:30.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Janet Andres, Ph.D. can be reached on 571-272-0867. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Elizabeth C. Kemmerer

Christina Borgeest, Ph.D.

ELIZABETH KEMMERER
PRIMARY EXAMINER